

Ocular Thrombosis after Stent-Assisted Coiling of a C7 (Paraclinoid) Internal Carotid Artery Aneurysm

A Report of Two Cases and Literature Review

KATSUNARI NAMBA¹, AYUHO HIGAKI², SHIGERU NEMOTO³

¹ Center for Endovascular Therapy, Division of Neuroendovascular Surgery, ² Department of Neurosurgery, Jichi Medical University; Tochigi, Japan

³ Department of Endovascular Surgery, Tokyo Medical and Dental University; Tokyo, Japan

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Summary

Stent-assisted coiling of paraclinoid aneurysms is widely performed in neurointerventional surgery. The most common adverse event related to this procedure is cerebral thromboembolism. However, reports on ocular thromboembolism are scarce.

We report our experience with two patients who developed ocular thromboembolism following Enterprise stent-assisted coiling of paraclinoid aneurysms. We then review the available literature for the possible pathomechanism of ocular thrombosis.

Ocular thromboembolism may be a risk of stent-assisted coiling when the stent traverses the orifice of the ophthalmic artery or the stent is placed in the C3 internal carotid artery. Further study is needed to clarify how to avoid this disabling complication.

Introduction

Thromboembolic complications are the most common adverse event in stent-assisted coiling of intracranial aneurysms¹. Published clinical studies describe the immediate incidence of cerebral thromboembolism to be between 2.8% and 5.4%¹⁻⁴. However, reports on ocular thromboembolism are scarce. Here, we describe two cases of ocular thromboembolism occurring after stent-assisted coiling of paraclinoid aneurysms not related to the ophthalmic artery.

Case Reports

Case 1

A 29-year-old woman was discovered to have an incidental left paraclinoid aneurysm on magnetic resonance imaging (MRI) and magnetic resonance angiogram (MRA) study during a workup for dizziness.

Subsequent cerebral angiography confirmed a left paraclinoid aneurysm measuring 4.8 × 5.0 mm with a 5.4 mm neck, accompanied by a 1.5 mm bleb (Figure 1A). The ophthalmic artery was independent of the aneurysm, and arose proximal to the aneurysm (Figure 1B). After discussing the benefits and risks with the patient, she consented to undergo stent-assisted coiling of the aneurysm.

The patient was started on aspirin 100 mg and clopidogrel 75 mg daily two weeks prior to the procedure. Drug-resistant testing was not available at our institution. Under general anesthesia, a bolus of 5000 units of heparin was administered intravenously, increasing the activated clotting time (ACT) from 130 seconds to 338 seconds. The ACT was maintained over 300 seconds throughout the procedure. Following catheterization of the aneurysm with an Excelsior SL-10 preshaped S microcatheter (Boston Scientific, Natick, MA, USA), a 4.5 × 28 mm Enterprise stent (Codman Neurovascular, Raynham, MA, USA) was deployed in the C1-4 portion of the internal carotid artery (ICA). The aneurysm was then coiled using the “jailed”

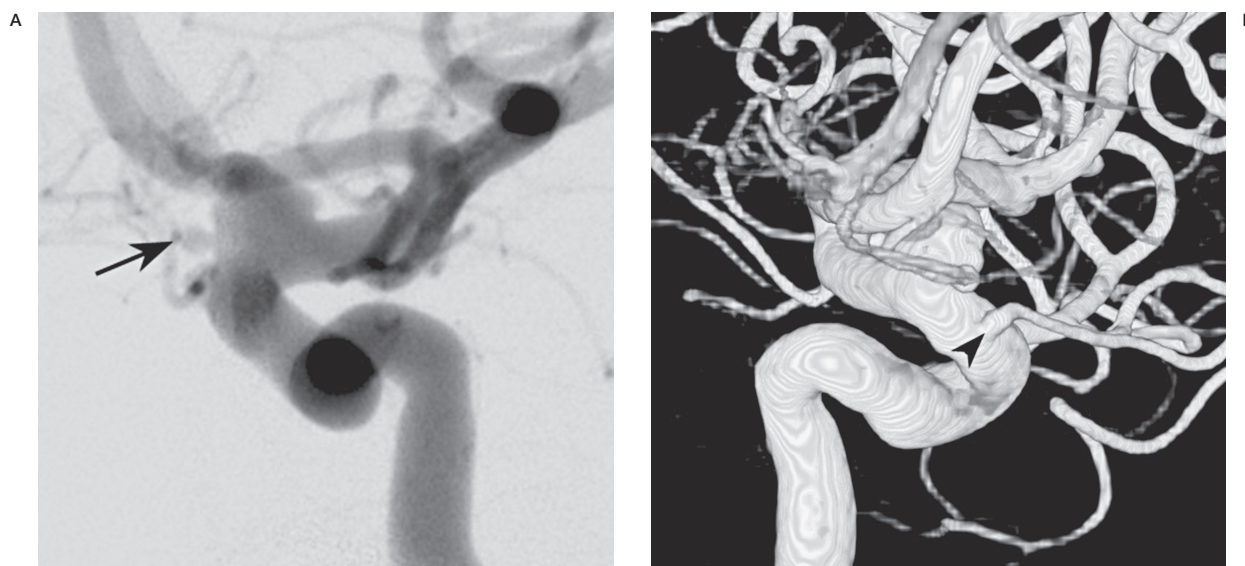


Figure 1 A) Left internal carotid artery angiogram in oblique view shows a wide-necked paraclinoid aneurysm with a bleb pointing anteriorly (arrow). B) An image of the left internal carotid artery reconstructed from the 3D rotational angiogram and demonstrating that the origin of the ophthalmic artery is separate from the aneurysm.



Figure 2 Post-procedure image of the left internal carotid artery reconstructed from flat-panel CT with contrast injection shows incomplete stent apposition to the vessel wall at the inner curve (arrows). Note the damage to the proximal end of the stent and the resulting malapposition to the vessel wall (arrowheads) caused by excessive force applied to the stent by microcatheter and guidewire.

microcatheter technique with HydroFrame 10 5 mm × 10 cm (Microvention Terumo, Tokyo, Japan) and Target 360 Ultra 3 mm × 8 cm (Stryker Neurovascular, Fremont, CA, USA) coils. Before achieving complete occlusion of the aneurysm, the catheter prematurely prolapsed from the aneurysm due to microcatheter

instability. Extensive attempts to re-catheterize the aneurysm were unsuccessful and the procedure was aborted. The post-procedure image obtained by flat-panel computed tomography (CT) with contrast agent injection revealed incomplete stent apposition at the proximal end and at the inner curve (Figure 2).

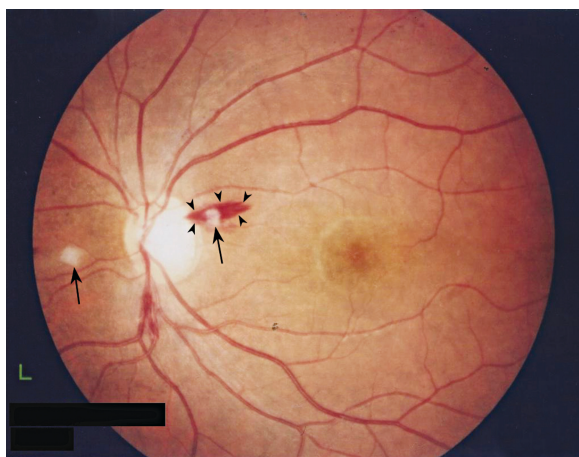


Figure 3 Funduscopy of the left eye on the 12th postoperative day demonstrates soft exudates (arrows) accompanied by a small hemorrhage (arrowheads), findings compatible with a small ocular thromboembolism.

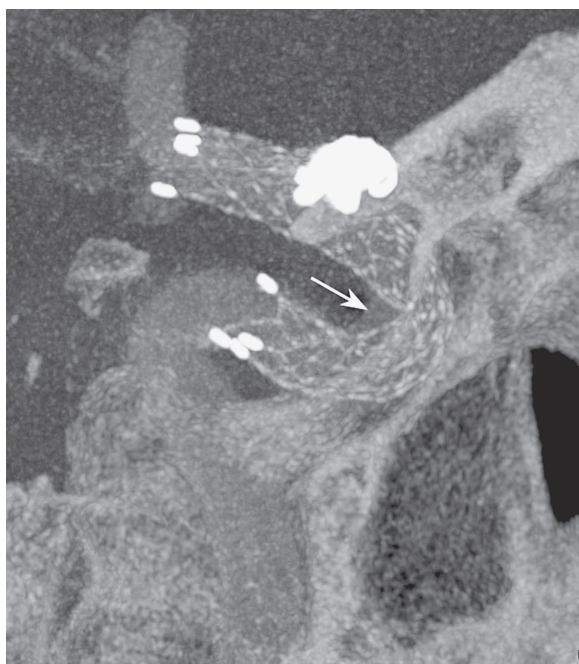


Figure 5 Post-procedure image of the right internal carotid artery reconstructed from flat-panel CT with contrast injection. Note the incomplete apposition of the stent to the vessel wall at the inner curve.

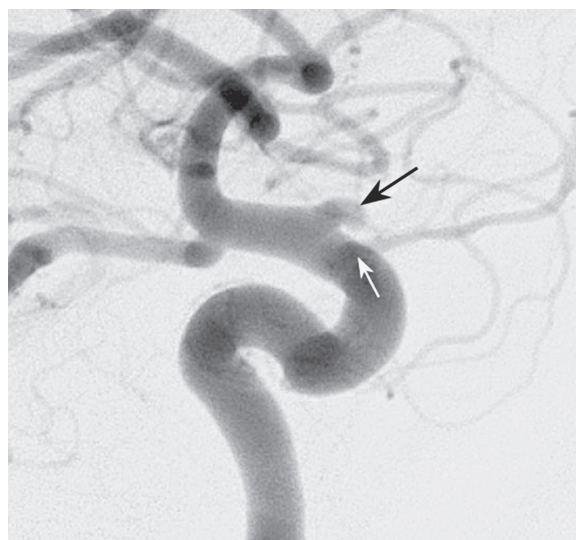


Figure 4 Right internal carotid artery angiogram in lateral view demonstrates a small, wide-necked paraclinoid aneurysm pointing anteriorly (black arrow). The origin of the ophthalmic artery is demonstrated by the double density where it originates (white arrow). The origin of the ophthalmic artery is unrelated to the aneurysm.

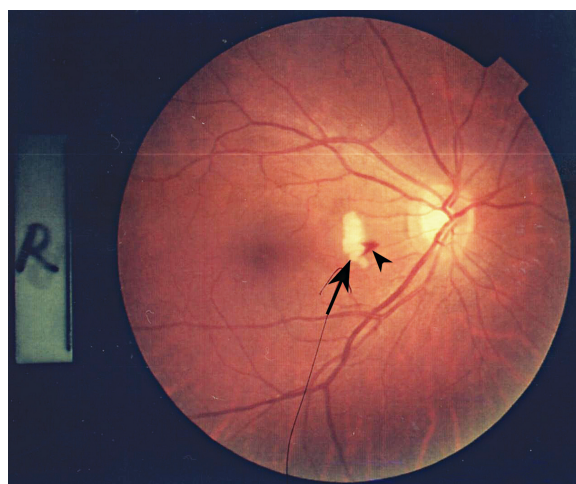


Figure 6 Right funduscopy picture obtained on the 11th postoperative day demonstrates a soft exudate (arrow) with a local hemorrhage (arrowhead), suggestive of a small thromboembolism.

The patient was maintained on heparin at 500 IU/h for 24 hours postoperatively. She was discharged without neurological deficits on the fifth postoperative day. However, soon after discharge, she recognized a visual impairment in the left eye. Ophthalmological examination on the twelfth postoperative day revealed a small

visual deficit in left lateral vision. Funduscopy performed on the same day demonstrated soft exudates accompanied by a small hemorrhage that was compatible with the diagnosis of ocular thromboembolism (Figure 3). MRI and MRA of the brain performed at the same time did not show cerebral ischemia or ICA occlusion.

Case 2

This 42-year-old woman presented to our institution because of a right paraclinoid aneurysm that was discovered during a workup for headaches. Cerebral angiogram revealed an anteriorly pointing right paraclinoid aneurysm measuring 1.7×3.2 mm with a 3.2 mm neck. The origin of the right ophthalmic artery was unrelated to the aneurysm, and was located proximal to the aneurysm (Figure 4). After discussing the benefits and risks of the stent-assisted coiling, she elected to undergo the procedure because of extreme anxiety over harboring the aneurysm.

The patient was started on aspirin 100 mg and clopidogrel 75 mg daily two weeks prior to surgery. Platelet function testing was not performed due to unavailability of the examination at our institution. Under general anesthesia, the patient was systemically heparinized with 5000 units of intravenous heparin. The ACT increased from 106 seconds to 267 seconds, and was maintained over 250 seconds throughout the procedure. The aneurysm was catheterized with an Excelsior SL-10 microcatheter and an intentional partial insertion of a Target 360 Nano 1.5 mm \times 3 cm coil (Stryker Neurovascular) was performed. This was followed by placement of a 4.5×28 mm Enterprise stent across the aneurysm. The first coil was then detached, and additional 360 Nano 1.5 mm \times 3 cm, and 2 \times 360 Nano 1 mm \times 3 cm coils were inserted using the “jailed” microcatheter technique. Postoperative flat-panel CT with contrast injection showed incomplete stent apposition at the inner curve (Figure 5).

The patient noticed blurred vision in the right eye that fluctuated in intensity on the third postoperative day. Ophthalmological examination of the right eye revealed concentric contraction of the visual field. Funduscopy performed on the eleventh postoperative day demonstrated a soft exudate with a small hemorrhage, compatible with the findings of ocular thromboembolism (Figure 6).

Discussion

We described two cases with ocular ischemic complication associated with stent-assisted coiling of paraclinoid aneurysms. Cerebral thromboembolism is widely recognized as a potential adverse event after stent-assisted coiling. To date, thromboembolic complications

have been reported to occur in the range of 2.8% to 5.4% following the procedure. However, to our knowledge, no detailed report describing ocular thromboembolism associated with stent-assisted coiling exists. The only description in the literature was found in the study by Mocco et al.⁴ Their multicenter register study using the Enterprise stent in 141 patients with 142 aneurysms, briefly described a patient developing a temporary “blind spot”. Unfortunately, no information on the location of the aneurysm or the procedure was given. Another report of treating 126 paraclinoid aneurysms, not specifically limited to stent-assisted coiling, one ocular thromboembolism and two amaurosis fugax were seen after the procedure⁵. The thromboembolism was related to the aneurysm being located at the carotid-ophthalmic junction and to the retreatment because of recurrence. In the two amaurosis fugax cases, details of the aneurysm location or the procedure were not provided. In our two cases of ocular thromboembolism, the aneurysms were independently located distal to the origin of the ophthalmic artery. Therefore, it is unlikely that local extension of the thrombus from the aneurysm caused the ocular complication.

The reason for ocular thromboembolism in stent-assisted coiling may be explained by two mechanisms: occlusion of the ophthalmic artery orifice by stent coverage, or stent thrombosis due to inadequate device apposition against the arterial wall. Occlusion of large side branches by stent placement has not been reported to the best of our knowledge. Furthermore, immediate or delayed occlusion of the ophthalmic artery or important side branches is rarely reported even when using a high surface coverage flow diverter stent in the paraclinoid carotid artery^{6,7}. Although this mechanism seems unlikely, we could not deny the possibility of branch occlusion because of lack of angiographic study at the occurrence of the symptom.

On the other hand, stent thrombosis may be the more likely mechanism. Heller et al. analyzed the correlation of stent apposition to the vessel wall and the occurrence of thromboembolic complications using 3 Tesla magnetic resonance diffusion-weighted imaging and time-of-flight angiography⁸. They found that incomplete stent apposition to the vessel wall was associated with increased ipsilateral hyperintense lesions on diffusion-weighted imaging. Thus, they concluded that incomplete stent apposition was associated with thromboembolic

complications in stent-mediated coil embolization of intracranial aneurysms. In our two cases, postprocedural flat-panel CT imaging showed incomplete stent apposition at the inner curve in both cases, and in case 1, at the proximal end due to stent damage. These may have served as the source of the stent thrombosis. In addition, fundus examination in our patients showed soft exudates. Soft exudate is a retinal degeneration caused by acute, local, and superficial ischemia. Because the retinal artery is an end artery, major occlusion of this vessel or the branch would have caused a larger lesion in the retina. The fundoscopic findings also support the hypothesis that the pathomechanism of the ocular ischemia was a small thromboembolism rather than a large vessel occlusion.

Stent design has an important impact at the paraclinoid ICA location because the stent is placed against the tortuous C3 portion. Open-cell design stent such as the Neuroform (Stryker Neurovascular, Fremont, CA, USA) has the advantage of better vessel wall conformability in tortuous vessel anatomy due to its flexible mechanism compared to the closed-cell design Enterprise⁸⁻¹¹. On the other hand, a disadvantage of the open-cell stent is the difficulty in navigating the system through tortuous vessels^{10,12}. By contrast, the closed-cell design stent is highly flexible and is advantageous in tortuous vessels. Other favorable features include the ability to reposition and the denser metal surface that confers better protection against coil herniation. Disadvantages of the closed-cell stent are stent migration, less vessel wall conformability, and the presumed thromboembolic complications^{8,10,11,13,14}. The previously mentioned study by Heller et al. demonstrated that the Enterprise showed significantly more incomplete wall apposition compared to the Neuroform stent⁸. While none (0 case out of 25) of the Neuroform stents showed incomplete stent apposition, 54.5% (18 cases out of 33) of the Enterprise stents resulted in incomplete stent apposition. Of note, 17 cases of incomplete Enterprise stent apposition were detected in the ICA, the same location as in our cases. The incomplete stent apposition that may have served as the cradle of thromboembolus formation in our cases may have been avoided had we used an open-cell design Neuroform stent. We may further suggest using an open-cell design stent in the C3 ICA location to avoid incomplete stent apposition, thereby lowering thromboembolic complications. However,

at present, we do not have clear answer to this question and await further investigation.

The retina may have some reasons for more vulnerability to ischemic insult than the brain, and subtle ischemia may lead to visual disturbance. For instance, the retina may have fewer collaterals compared to the cerebral circulation. There also may be differences in cellular tolerance to ischemia or in the threshold for symptom awareness¹⁵. Therefore, judicious usage and selection of stents may be necessary in the paraclinoid carotid artery.

There are several limitations to this report. First, there is no information on antiplatelet treatment. Although our patients were started on a double antiplatelet regimen two weeks before the procedure, neither patient was tested for antiplatelet efficacy. Inadequate antiplatelet inhibition may have played a role in the thrombosis. Second, in case 1, stent damage at the proximal end caused markedly incomplete apposition and this may have promoted thrombus formation. However, whatever the mechanism of the incomplete apposition may be, ocular thrombosis was caused by placing a stent in proximity to the origin of the ophthalmic artery. Third, since we used a closed-cell Enterprise stent, we do not have any answer to whether an open-cell design stent could decrease the incidence of such complications. Further studies are required to answer this question. Finally, the small number of cases precludes any definitive conclusions drawn from our report. Further accumulation of experience is needed to clarify the cause and incidence of this complication. However, although rare, we would like to emphasize that ocular thrombosis may occur as a consequence of stent-assisted coiling, and that this complication should attract wider recognition.

Conclusion

We described two cases of ocular thrombosis occurring after stent-assisted coiling of paraclinoid aneurysms. This complication has not been described as a potential risk of stent-assisted coiling, and may require more attention. For paraclinoid aneurysms, the addition of funduscopy as a follow-up examination may be useful to increase the sensitivity for thromboembolic complications. Although we do not have solid evidence, the thrombosis may have been caused by stent thrombosis.

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Katsunari Namba, MD
 Center for Endovascular Therapy
 Division of Neuroendovascular Surgery
 Jichi Medical University
 3311-1 Yakushiji
 Shimotsuke, Tochigi 329-0498, Japan
 Tel.: +81-285-58-7373
 Fax: +81-285-44-5147
 E-mail: knamba@jichi.ac.jp